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Unusual causes, including paragonimiasis,[427] are described.[350] The clinical context in which the effusion occurs is so characteristic that pseudochylothorax rarely causes diagnostic confusion with a true chylothorax. Imaging may also help differentiate between these entities, since true chylothorax is rarely associated with pleural thickening, loculation, or calcification.

HEMOTHORAX

Hemothorax usually results from trauma.[428] However, on occasion it occurs in other conditions (Box 15.9). The natural history of hemothorax depends in part on the source of the bleeding. Low pressure bleeding from the lung tends to stop spontaneously because the pleural fluid compresses and collapses the lung. High pressure bleeding from systemic vessels is less susceptible to the tamponade effect of pleural fluid,[448] and the bleeding may be rapid and persistent with the formation of a tension hemothorax.[429] In the context of trauma other causes of rapidly accumulating pleural fluid should be considered, including ruptured esophagus, ruptured thoracic duct, traumatic subarachnoid pleural fistula,[449] and iatrogenic causes, particularly venous perforation from line placement.

Box 15.9 Causes of hemothorax

Trauma
Open
Closed (with or without fracture)
Iatrogenic[431]

Infection
Varicella[432]

Coagulopathy
Hemophilia[433]
Anticoagulants[429,434,435]

Vascular abnormality
Arteriovenous malformation[436]
Dissecting aortic aneurysm
Atherosclerotic aneurysm[437]

Rib exostosis[438]

Neurofibromatosis with pregnancy[130]

Pulmonary and pleural neoplasms[437,439]

Extramedullary hemopoiesis[440,441]

Pneumothorax[430,442]

Catamenial hemothorax (endometriosis)[443,444]

Idiopathic[445–447]

In the acute state nothing on the chest radiograph distinguishes hemothorax from other collections of pleural fluid. However, on CT a hemothorax may show areas of hyperdensity (see Fig. 15.14).[450] With clotting of the blood, loculation tends to occur and fibrin bodies may form.[59,430] Hemothorax may eventually organize and cause massive pleural thickening (fibrothorax), necessitating decortication, a complication that can be avoided by early evacuation of the pleural space.

PLEURAL THICKENING

Pleural thickening can be localized or diffuse, and usually represents the organized end stage of a variety of active processes, particularly infective and noninfective inflammation, hemothorax, and asbestos- and drug-related disease (see Box 15.10). It is virtually always present after thoracotomy and pleurodesis,[451] and may follow irradiation. The most common causes are probably hemothorax, bacterial infection, and tuberculosis.[5] Particularly marked pleural thickening is seen following tuberculosis or irradiation. Identification of diffuse pleural thickening is important because it is commonly associated with significant restrictive physiologic impairment. It can be important to try to distinguish between diffuse pleural thickening, which may be due to a large variety of causes, and localized pleural plaques which are usually related to asbestos exposure (see p. 1043). On the chest radiograph, diffuse postinflammatory pleural thickening almost always involves the costophrenic sulci (Fig. 15.44), while pleural plaques present with localized areas of soft tissue density along the chest wall. The presence of bilateral abnormality favors asbestos related disease.[10]

Box 15.10 Causes of pleural thickening

Infection
Tuberculosis
Empyema

Hemothorax

Asbestos exposure

Surgery or pleurodesis

Radiation

Malignancy
Metastasis
Mesothelioma
Leukemia
Lymphoma

The radiographic changes of diffuse pleural thickening are more commonly unilateral and consist of soft tissue shadowing, characteristically in the more dependent lateral and posterior parts of the chest. There may be radiographic signs of ipsilateral rib enlargement in patients with chronic (particularly tuberculous) pleural disease.[452] Blunting of the costophrenic angle is common and is often angular, distinguishing it from the more smoothly curvilinear pleural fluid. Decubitus radiographs and ultrasonography are particularly helpful in making this distinction. En face, extensive pleural thickening gives a veil-like opacity that has no clear margins and crosses known pulmonary boundaries. Tangentially, it appears as a soft tissue density immediately inside and parallel to the chest wall, sharply marginated on its inner aspect and fading into the soft tissues of the chest wall laterally. Such pleural thickening can extend into and thicken fissures.

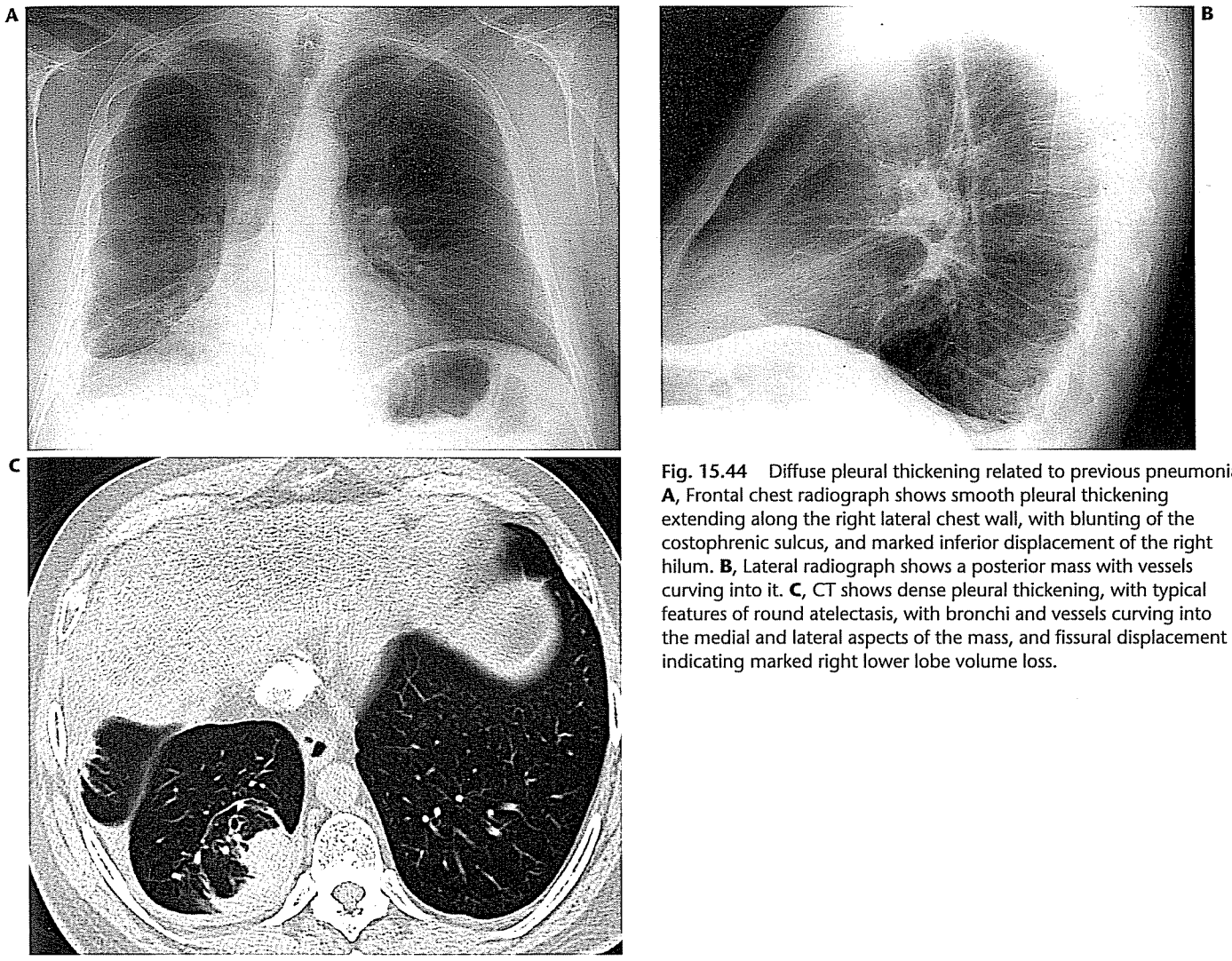


Fig. 15.44 Diffuse pleural thickening related to previous pneumonia. **A**, Frontal chest radiograph shows smooth pleural thickening extending along the right lateral chest wall, with blunting of the costophrenic sulcus, and marked inferior displacement of the right hilum. **B**, Lateral radiograph shows a posterior mass with vessels curving into it. **C**, CT shows dense pleural thickening, with typical features of round atelectasis, with bronchi and vessels curving into the medial and lateral aspects of the mass, and fissural displacement indicating marked right lower lobe volume loss.

On ultrasound, pleural thickening produces a homogeneously echo dense layer subjacent to the chest wall, but it cannot be reliably detected unless it is about 1 cm or more thick.[86] There is no posterior echo enhancement, but this is often difficult to assess because the soft tissue–lung interface is normally so reflective.

On CT, pleural thickening is detected as a layer of soft tissue opacity lying at the chest wall–lung interface. It can be detected almost as well with conventional CT as with HRCT, though the latter is more sensitive in assessing asbestos related plaques.[453] In addition, HRCT may sometimes clarify equivocal findings on conventional CT.[454] On HRCT, pleural thickening is best assessed inside ribs where there should be no discernible soft tissue; exceptions to this "rule" are discussed on page 40.[455] Paravertebrally any thickening of the normally insignificant pleural line is abnormal. HRCT is very sensitive and can detect thickening on the order of 1–2 mm. The extrapleural fat layer, which is normally absent or relatively thin, thickens in chronic pleural disease, particularly with chronic empyema,[455–458] making appreciation of pleural thickening easier. When this fat has higher density than usual, it suggests that there is active inflammation in the pleural space.[459] Both the distribution and morphology[89] of diffuse pleural thickening are helpful in identifying a cause. In one study the specificity of various CT signs in differentiating a malignant from a benign pleural process was evaluated. The four most useful signs of malignancy (with specificities) were circumferential thickening (100%), nodularity (94%), parietal thickening >1 cm (94%), and mediastinal pleural involvement.[454] Early experience suggests that positron emission tomography (PET) with ^{18}F-fluoro-deoxyglucose (^{18}F-FDG) may have a role in the differentiation between malignant and benign pleural disease.[460] In a study of 23 patients, ^{18}F-FDG PET correctly identified all 16 cases of malignant pleural infiltration; there was intense uptake of ^{18}F-FDG in 14 of 16 cases (Fig. 15.45). Seven of nine benign lesions were characterized by an absence of tracer uptake but there was moderate uptake in two patients, one with a parapneumonic effusion and one with tuberculous pleurisy.[460] Another study found that MRI was more accurate than CT in diagnosis of malignant pleural disease: malignant disease was associated with increased signal intensity on T2-weighted images, and with enhancement following gadolinium administration.[461]

Pleural thickening, particularly when generalized, is often correctly dismissed as an inactive residuum. Care, however, must

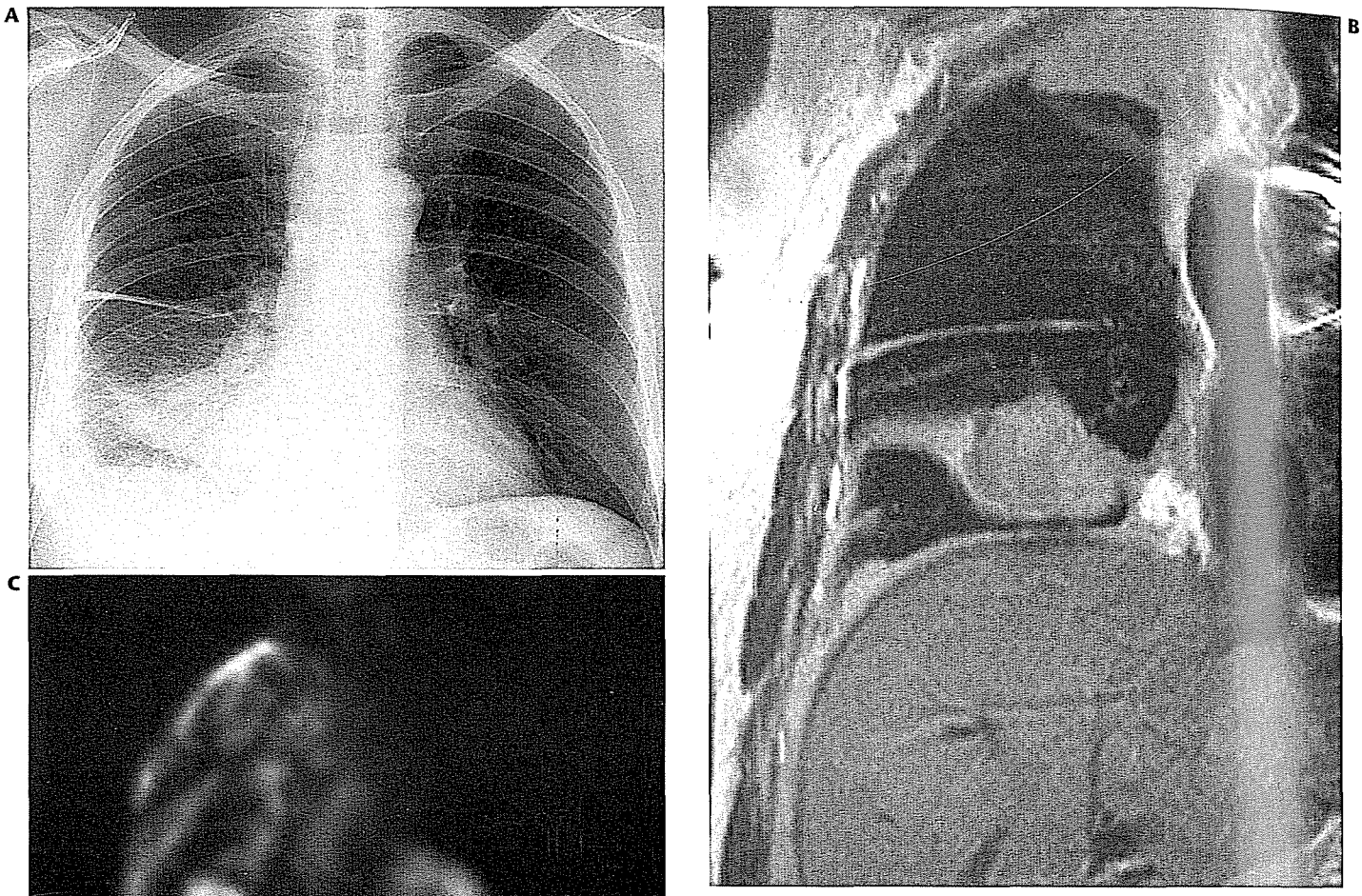


Fig. 15.45 Activity of PET in malignant pleural disease due to mesothelioma (same patient as in Fig. 15.32). **A**, Chest radiograph shows circumferential pleural thickening. **B**, Coronal MRI confirms the presence of circumferential pleural thickening extending into the costophrenic sulci, with a mass in the major fissure. **C**, Coronal PET image obtained with ^{18}F-fluoro-deoxyglucose shows circumferential increase in metabolic activity, extending into the fissures.

be taken to distinguish it from various active processes, some of which are neoplastic. Although a number of these conditions tend to give plaquelike, nodular, or irregular shadowing (Fig. 15.45), they occasionally closely resemble simple inactive pleural thickening. Disorders to consider include mycetoma related pleural thickening,[462] mesothelioma, diffuse pleural metastases (e.g. from thymoma[463]), leukemia,[464] lymphoma, and Wegener granulomatosis. It should also be remembered that a thick pleural peel occurring following empyema will usually decrease progressively over the subsequent 12 weeks.[465]

There are several uncommon syndromes of idiopathic bilateral pleural thickening. In the entity of cryptogenic bilateral fibrosing pleuritis there is widespread thickening of the pleura preceded by effusions (Fig. 15.46).[466,467] Patients may have an elevated erythrocyte sedimentation rate, restrictive lung function, and evidence of rounded atelectasis on CT.[466] Another uncommon idiopathic syndrome is associated with progressive apical pleural and subpleural fibrosis, with restrictive physiology.[468] This syndrome may be familial,[469] and may be associated with renal tubular acidosis.[470]

Mimics

Pleural thickening must be differentiated from apical pleural caps and extrapleural fat. The apical pleural cap, though it looks like pleural thickening, is usually due either to extrapleural fat or to subpleural fibrosis. Older publications refer to serratus

Fig. 15.46 Cryptogenic bilateral fibrosing pleuritis. Prone CT image shows bilateral smooth pleural thickening with bilateral lower lobe volume loss and parenchymal scarring.

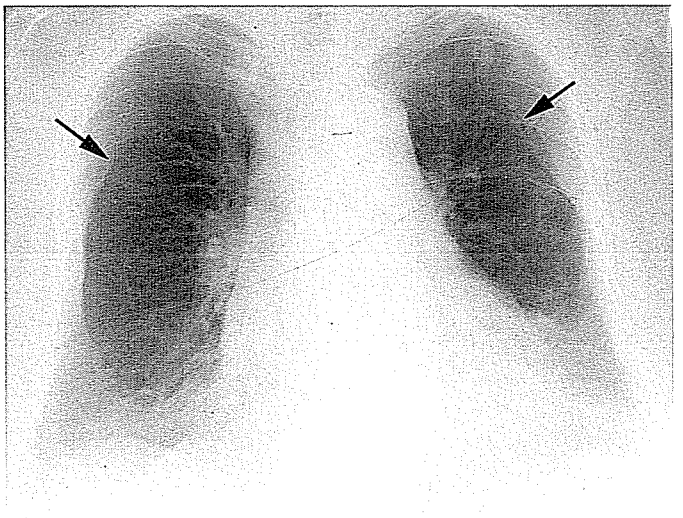


Fig. 15.47 Extrapleural fat in a patient treated with steroids. Chest radiograph shows lobulated smooth thickening along the chest wall bilaterally, extending to the lung apices, and extending. Symmetric curvilinear interfaces in the upper chest (arrows) are due to invagination of fat into the major fissures bilaterally.

anterior shadows and rib companion shadows as mimics of pleural plaques on chest radiographs.[471,472] These are not discussed here because it is very rare for the serratus anterior muscle or ribs to be so sharply outlined by air as to produce a radiographically distinct interface.

Extrapleural fat

Extrapleural fat may generate confusing shadows that can resemble generalized pleural thickening or plaques (Fig. 15.47).[473,474] The distribution varies from patient to patient. Sometimes the fat is widely distributed, mimicking a pleural peel. At other times, it is localized and develops particularly over the fourth to eighth ribs between the anterior axillary line and the rib angles.[473] Excess thoracic fat is more common in obese patients, and associated with mediastinal lipomatosis, but may also be found in thinner individuals. Extrapleural fat may be distinguished from pleural thickening by its symmetry and its undulating outline, often extending to the lung apices but sparing the costophrenic sulci.[473] If doubt remains, CT will readily differentiate thickening from fat, but this distinction is not usually clinically important.[473]

Apical pleural cap

An idiopathic apical pleural cap is an irregular, usually homogeneous, soft tissue density that is found at the extreme lung apex (Fig. 15.48).[475] The lower border is usually sharply marginated and may be smoothly curvilinear, tented, or undulating.[476] Caps are usually <5 mm thick, but the width is variable. In two series caps were about as common unilaterally (11 and 7%) as bilaterally (11 and 12%).[476,477] When bilateral the caps were usually asymmetric. The frequency of occurrence increases with age: 6.2% up to 45 years of age and 15.9% over

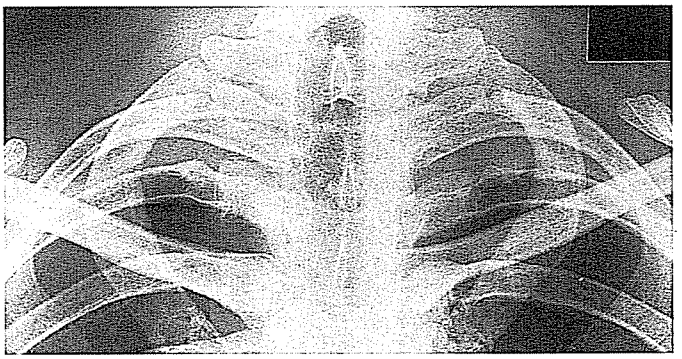


Fig. 15.48 Apical pleural caps. Symmetric soft tissue opacities are projected under both second ribs. They are slightly atypical for pleural caps, being thicker (1 cm) than usual, with some irregularity of their lower margins. The appearance of pleural caps is quite variable.

45 years of age.[475] The opacity is formed by an apical subpleural scar that is nonspecific and unrelated to tuberculosis.[478]

In contrast to the smooth or undulating outline of the apical cap on the chest radiograph, CT usually shows a subpleural irregular linear abnormality, consistent with the pathologic findings of dense subpleural fibrosis, and sometimes difficult to distinguish from a spiculated lung cancer (Fig. 15.49). Indeed, Yousem[479] presented a series of 13 such cases which had been resected because of suspected lung cancer.

The differential diagnosis of an idiopathic apical cap includes nongranulomatous and granulomatous (tuberculous, fungal) infection, radiation pleuritis, lymphoma, pleural and extrapleural neoplasms, extrapleural hematoma, prominent subclavian artery, mediastinal lipomatosis,[480] and apicolateral extrapleural fat.[481] A HRCT study has shown, somewhat surprisingly, that the bulk of the apical "pleural" opacity

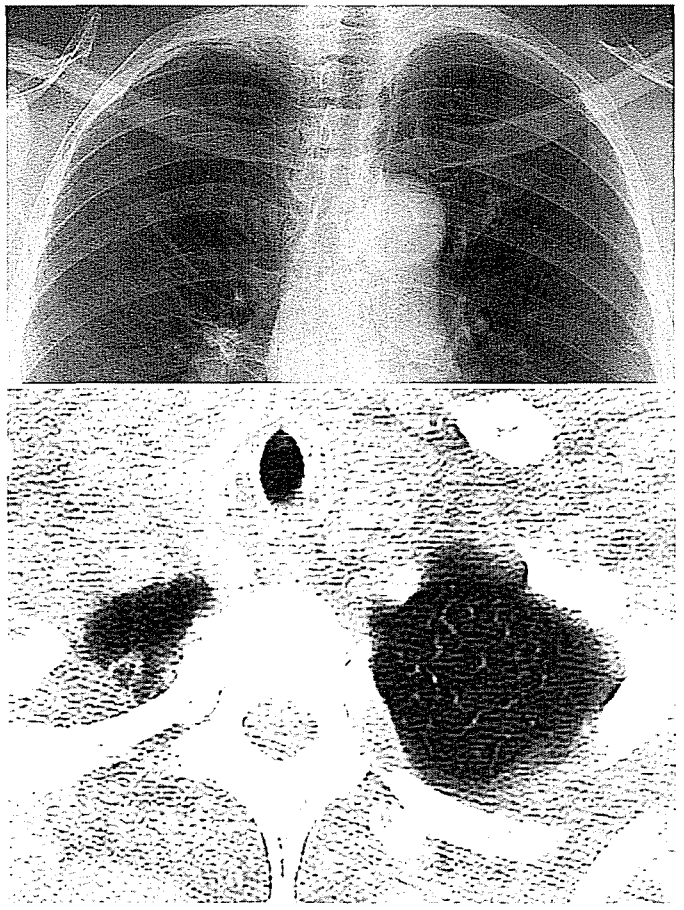


Fig. 15.49 Apical pleural scar. **A**, Chest radiograph shows asymmetric soft tissue thickening at the right apex. **B** and **C**, CT shows irregular subpleural density, with spiculation mimicking lung cancer. The patient was followed for 2 years without evidence of progression.

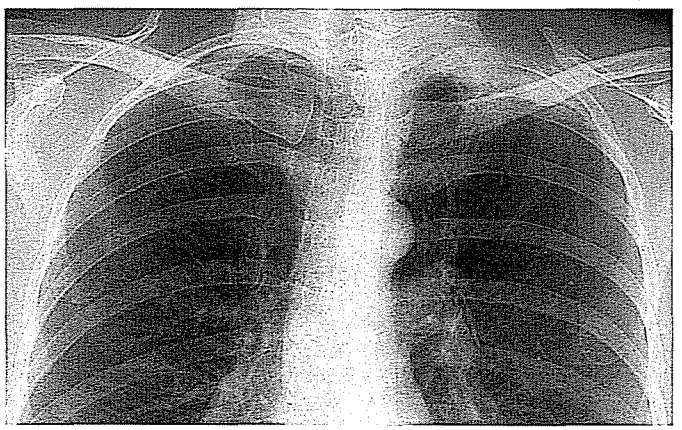


Fig. 15.50 Pancoast tumor. Chest radiograph shows apical pleural thickening associated with destruction of the posterior left second rib.

associated with previous tuberculosis is caused by a thickened (up to 25 mm) layer of fat between visceral pleura and the endothoracic fascia-innermost intercostal muscle stripe.[482] This may be related to contraction of the upper lobe, with filling of the extrapleural space by fat. The most important differential diagnosis is with Pancoast tumor, which should be suspected if there is marked asymmetry or nodularity of apical pleural thickening, if the patient has local pain, and particularly if there is underlying bone destruction (Fig. 15.50).[480]

RADIOLOGIC APPEARANCE FOLLOWING PLEURODESIS

The most common indications for pleurodesis are recurrent pneumothorax, and malignant pleural effusion. The perfect agent for pleurodesis has not yet been found, but talc is increasingly used in place of chemical agents such as tetracycline or bleomycin, because of its higher success rates and lower rate of local and systemic symptoms.[336,483,484] Talc may be administered either as an aerosolized powder at thoracoscopy,[485,486] or as a slurry via large- or small-bore chest tubes at the bedside.[336,487,488]

After pleurodesis, the pleural space usually undergoes a phase of organization, with pleural thickening and loculations evident on chest radiograph; about 60% of patients have radiographically visible pleural thickening at longterm follow up.[451] On CT obtained after talc pleurodesis, the pleural space usually reveals variable degrees of pleural thickening and nodularity, often with a residual loculated effusion. High-attenuation areas representing talc deposits may mimic pleural calcification (Fig. 15.51).[489] The use of talc as a pleurodesis agent remains controversial, mainly because of recurrent reports of acute respiratory distress syndrome or lung edema occurring after talc pleurodesis.[485,490–492] Although expert opinion favors its use, the search for a better agent continues.

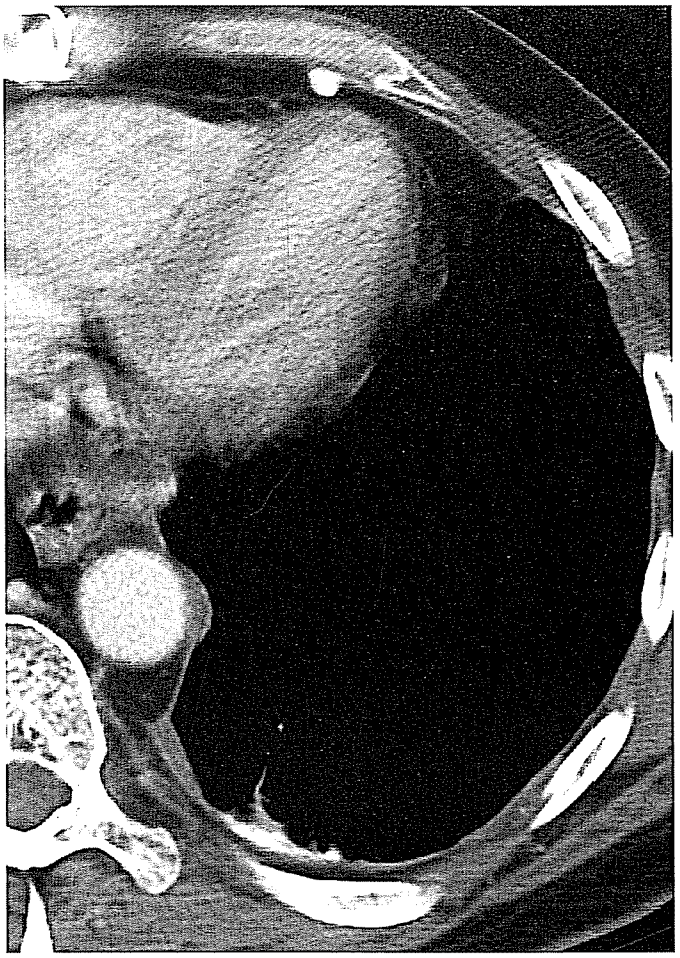


Fig. 15.51 CT appearances following talc pleurodesis. CT in a patient who had aerosolized talc pleurodesis for a left-sided mesothelioma shows a nodular area of hyperattenuation along the left posterior chest wall. A further nodular collection of talc is seen anterior to the heart.

Box 15.11 Causes of pleural calcification

Infection
Tuberculous empyema
Nontuberculous empyema

Hemothorax

Mineral inhalation
Asbestos (including tremolite talc)
Mica
Zeolites

Miscellaneous
Chronic pancreatitis[520]
Chronic hemodialysis[521]
Calcified metastasis
Alveolar microlithiasis[522,523]

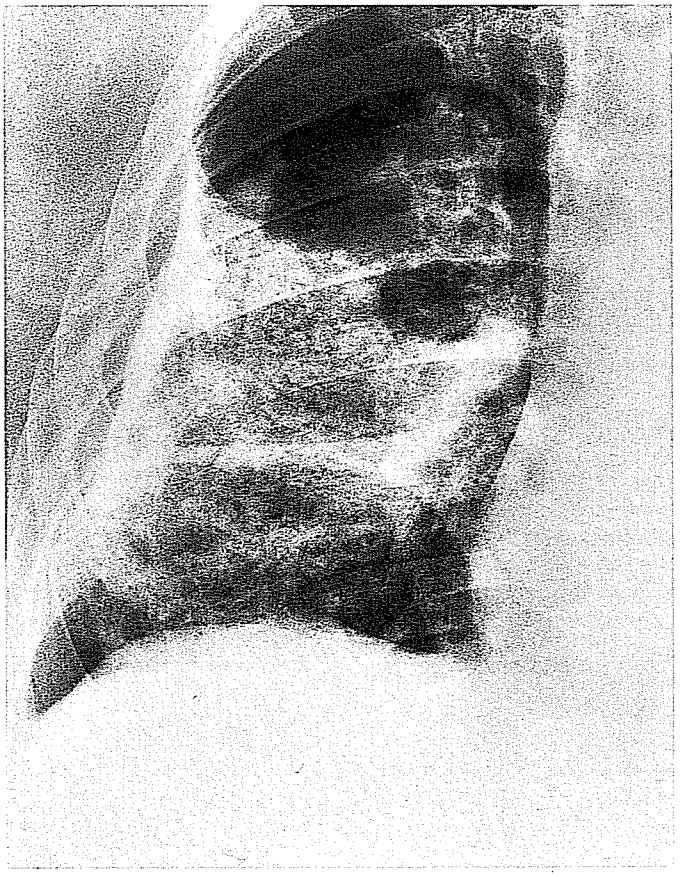


Fig. 15.52 Pleural calcification. Localized view of right middle and lower zone shows sheetlike calcification. Laterally, where calcification is tangential to the x-ray beam, it is dense and homogeneous, but medially – where it is seen en face – it is more broken up and nodular. There is a 1 cm thick band of noncalcified pleural thickening along the lateral chest wall.

PLEURAL CALCIFICATION

Virtually any process that can cause pleural thickening can be responsible for later pleural calcification,[493] but in practice calcification is usually due to infection, hemorrhage, or asbestos exposure. The recognized causes are listed in Box 15.11.

Calcification in asbestos inhalation and related conditions is morphologically characteristic,[494] and is considered on page 451. Calcification following infection and hemorrhage generally cannot be distinguished from each other. Such calcification is usually unilateral and varies from barely detectable to massive (Fig. 15.52). In the latter circumstance it becomes sheetlike and, reflecting the gravitationally determined distribution of the preceding pleural fluid, is often concentrated posterolaterally.[493] En face it appears as a hazy veil-like opacity, but in profile it is dense and linear, often parallel to the inner chest wall. The calcification in old empyemas occurs in both visceral and parietal pleura.[457,495] Sometimes these calcified layers are separated, an observation that can be made on radiographs or more easily on CT (Fig. 15.53).[457] In a series of 140 calcified fibrothoraces, 15.7% had a persistent effusion that was sandwiched between layers of thickened calcified pleura and was demonstrable on CT by virtue of its attenuation, location, homogeneity, and failure to enhance.[457] This can be suspected from the plain radiograph with pleural thickening of >2 cm and a double layer of calcification.[457] Active infection of these loculated collections is manifest by expansion of the pleural opacity and development of an air–fluid level signifying a bronchopleural fistula.[495]

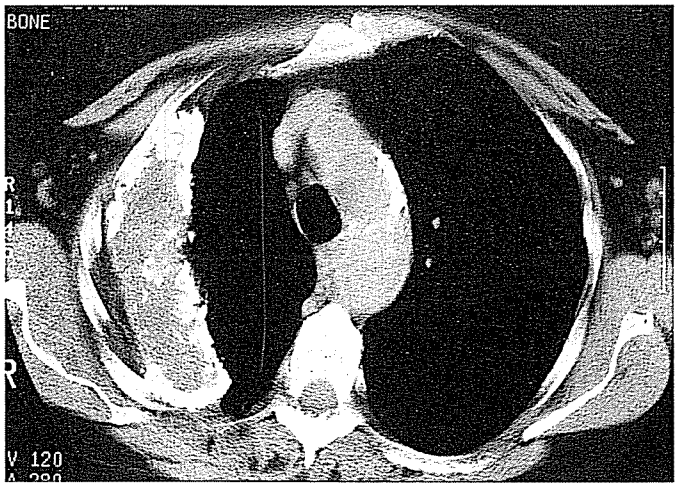


Fig. 15.53 Pleural calcification – old tuberculous empyema. Lenticular pleural opacity shows soft tissue density centrally and is marginated by heavy calcification both in the visceral and parietal pleura.

Occasionally postempyema calcification in the pleural space is manifest as a milk of calcium collection. These are often lenticular in shape and surrounded by mildly thickened pleura. On CT they are high density (200–300 HU) and typically homogeneous.[479]

THORACIC SPLENOSIS

Thoracic splenosis occurs when tissue from a traumatized spleen crosses an injured diaphragm and proliferates within the left hemithorax.[497] It is an uncommon condition[498]; in one prospective review there was evidence of splenic tissue within the thorax in only three of 17 patients who had sustained combined splenic and diaphragmatic injury.[499] Tissue from a traumatized spleen crosses an injured diaphragm and proliferates within the left thorax.[497] The resulting pleural nodules are often multiple and usually <3 cm in diameter, but may be up to 7 cm.[497] The nodules are implanted on parietal or viscera pleura, including fissures.[500]

On radiologic study, lesions of splenosis usually appear as pleural masses of soft tissue attenuation (Fig. 15.54), but they may appear intraparenchymal both on conventional radiographs and CT.[497] It is likely that the majority of apparently intrapulmonary lesions have pleural contact, though possibly some have been implanted in a lung laceration rather than on the pleural surface. On CT, the lesion may be lobulated or smooth and of soft tissue density.[499] On T1- and T2-weighted MRI the masses have been shown to be isointense with paraspinal muscles and subcutaneous fat, respectively.[499] Should the spleen have been removed at the time of trauma, the absence of Howell-Jolly bodies in a blood film would suggest persisting ectopic splenic activity.

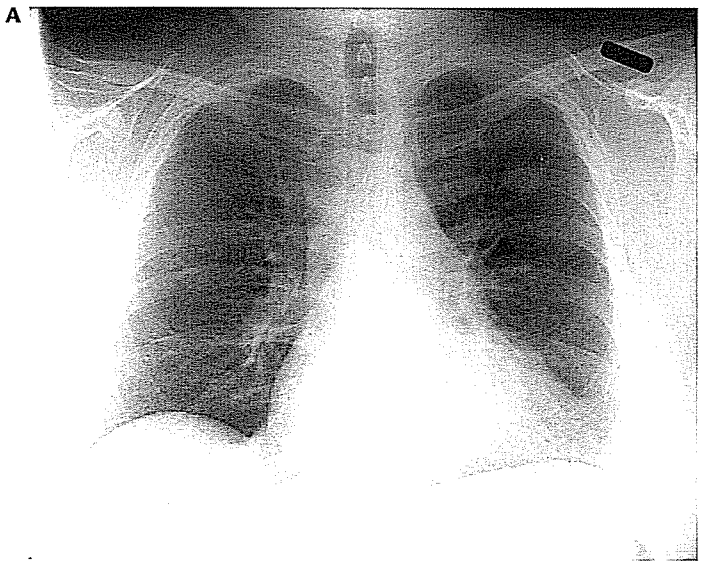
The diagnosis may be confirmed with scintiscans using ^{99m}Tc-sulfur colloid, ^{99m}Tc-labeled heat damaged erythrocytes, or ^{111}In-labeled platelets, all of which are taken up by the ectopic splenic tissue.[497,501–503]

PNEUMOTHORAX

Traditionally, pneumothorax is divided into spontaneous and traumatic types. The most common causes in adults are listed in Box 15.12. Only spontaneous pneumothorax is discussed in this chapter, apart from a brief consideration of pneumothorax associated with mechanical ventilation.

Primary spontaneous pneumothorax

A pneumothorax occurring without an obvious precipitating traumatic event is termed *spontaneous*; if, in addition, the individual is apparently healthy, the pneumothorax is termed



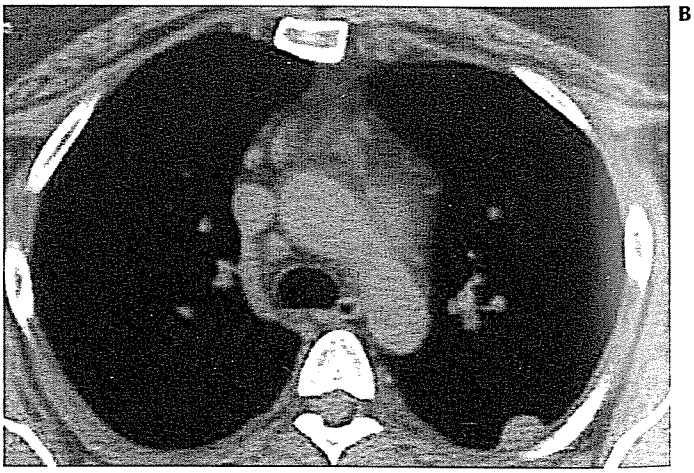


Fig. 15.54 Thoracic splenosis in a patient with a history of previous trauma. **A**, Chest radiograph shows a well-defined soft tissue mass in the left upper chest. **B**, CT confirms a posterior pleural mass. Splenosis was confirmed by sulfur colloid scan.

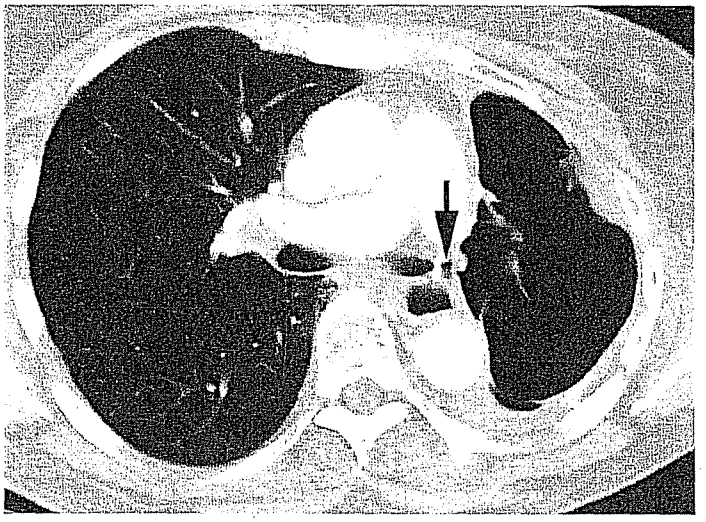


Fig. 15.71 Central bronchopleural fistula in a 70-year-old woman following left pneumonectomy for a bronchial carcinoma. CT shows a large amount of air in the pneumonectomy space. Air leads from the bronchial stump to the pneumonectomy space (arrow).

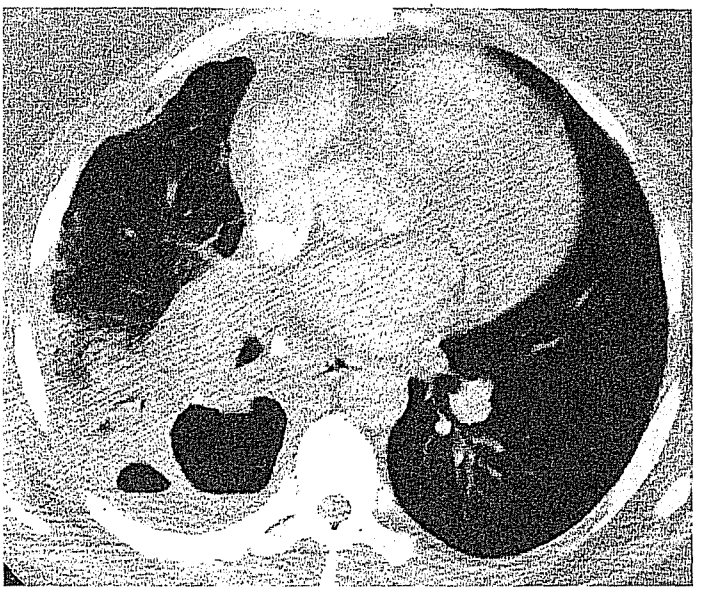


Fig. 15.72 Peripheral bronchopleural fistula in a 37–year-old female following wedge resection of the superior segment of the right lower lobe. There is air in the pleural space, but the fistula is not directly demonstrated.

Bronchopleural fistulas complicating infections (Fig. 15.62) are considered in the discussion of empyema (see p. 203). Postsurgical fistulas are considered here. They occur with a frequency of about 2.5–3%[746,747] and usually develop within 2 weeks of surgery. They should be suspected with the postoperative development of fever, hemoptysis, cough (especially if productive of a large amount of brown sputum), and a persistent large air leak from the pleural drains. Postoperative bronchopleural fistulas are usually associated with infection, and are much more common after surgery performed for pulmonary infections such as tuberculosis.[748]

The chest radiographic signs of bronchopleural fistula following recent pneumonectomy are: (1) a sudden increase in the amount of air in the pneumonectomy space, or in the adjacent chest wall; (2) a decreased amount of fluid; (3) loss of the normal mediastinal shift toward the operated side; and (4) sometimes a contralateral parenchymal opacity due to aspiration of fluid from the pneumonectomy space. Occasionally, unchanged persistence of an airspace following pneumonectomy indicates a fistula. This happens when the residual space is surrounded by pleural fibrosis and scarring so that it cannot change shape.[746] Extensive scarring may also prevent the mediastinal shift sign from being seen.[749] It is not uncommon for radiographic signs (increasing pleural air) of bronchopleural fistula to appear in otherwise well patients who go on without complication or interference to successful obliteration of the pleural cavity.[750] This is ascribed to a flap valve type of fistula that is self healing. Delayed bronchopleural fistula, occurring after air has been eliminated from the pneumonectomy or lobectomy space, is signaled by the reappearance of air in the pleural space (Fig. 15.71).

A fistula may be detected with ^{133}Xe lung scintigraphy in the washout phase.[751] However, it usually cannot be demonstrated with DTPA aerosol. Injection of water soluble contrast into the relevant bronchus, or into the pleural space may occasionally be helpful. CT can be helpful in depicting the anatomic details of a fistula, particularly in a peripheral bronchopleural fistula which cannot be directly visualized at bronchoscopy.[741,743] Although CT will directly visualize a peripheral bronchopleural fistula in only 30–50% of cases, it will show a probable cause of the bronchopleural fistula (e.g. peripheral cavity, bulla) in most of the others.[742,743] Use of thin sections through areas of suspected fistula may be helpful in directly identifying the site of leakage.[743] Postoperative peripheral bronchopleural fistulas (Fig. 15.72), or those related to bullae, are less likely to be demonstrated than other types of fistulas.[742,743]

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